- 47. Speers M.A. et at Occupational exposures and brain cancer mortality: a preliminary survey of East Texas residents. American Journal of Industrial Medicine Vol 13 pp 629 38 1988
- 48. Types T et al., Incidence of cancer in Nowegian Workers potentially exposed to electromagnetic fields. American Journal of Epidemiology Vol 136 pp 81 88 1992.
- 49. Bielec M. Experimental and epidemiological investigations on risk of cancer in subjects exposed for a long time to microwave and radiofrequency fields WIHE Warsaw 1985
- 50. Swerdlow A.J. Epidemiology of eye cancer in adults in England and Wales 1962 -77. American Journal of Epidemiology Vol 13 pp 294 300 1983
- 51. Project Pandora Papers made public released by US State Dept as Project Pandora cited as USDD-PP and Microwave US USSR Vols 1-9.

52."ibid

53 Stenick op cit

Bach S A., Baldwin M., Lewis S; Some Effects of Ultra Righ Frequency Energy on Primate Cerebral Activity. TS-111 1959 p 83

54. Ibid

55. Ibid

56, ibid

56a ibid

56b Allen S G, Berhardt, C M H, Driscoll M, Gandolfo, Mariutti R, Matthes R, McKinley A F, Steinmetz M, Vecchia P, Whillock M; Proposals for basic restrictions for protection against occupational exposure to electromagnetic non Honising radiations. Physica Medica Vol VII 1991 p 83

- 57. More Protection From Microwave Radiation Hazards Needed.
 Report by the Comptroller General of the United States US General Accounting Office Nov 1978.
- 58. Danikvskyiy V Y ., Studies of the physiological effects of electricity at a distance: Parts 1 & 2 1900 & 1901
- 59. Vernadskyjy V L., The Biosphere. 1926
- 60. Patzild IZ., Hochfrequenziechn., 1930 Vol 36 p 85

- 61. Mirutenko VI., The thermal effect and certain problems in the dosimetry of the pulsed superhigh frequency (microwave) electromagnetic field
- 62. Deichmann W. B., Stephens FH., Microwave Radiation of 10mw/cm2 and factors that influence biological effects at various power densities.

 Industrial Medicine and Surgery 1961 Vol 30 pp 221 223.
- 63. Petrov I R ed Influence of microwave radiation on the organism of man and animals
- 64. Thomas T L., Stolley P D., Stemhagen A. Foutham E T E., Bleeker M L., Stewart R A., Hoover R N., Brain Tumour mortality risk among men with electrical and electronics jobs: a case control study. J. Natl Cancer Inst 79 223 1987.
- 65. Kholodov Y A The effect of electromagnetic and magnetic fields on the central nervous system. pp 213-216 Translated by NASA 1967.
- 56. Thid
- 67. Ryan P., Lee M W., North B., McMicheal A J., Risk factors for tumours of the brain and meninges: results of the adelaide brain tumour study. Int J Cancer 51, 20-27 1992
- 68. Kholodov Y A op cit
- 69. Libenza P., Short and Ultra short waves
- 70. Suponitskaya F M., Arkh patol. anat. 1937, 3,2 p 113
- 71. Petrov op cit
- 72. ibid
- 73. ibid
- 74. ibid
- 75. Bychkov M.S., Electrographic Data on the effects of very weak microwaves at the level of the midbrain reticular formation Hypthalamus Cereblar Cortex Level, in Gordon., Biological Effects of Radiofrequency Electromagnetic Fields JPRS 63321 1974 p 75-86
- 76. Gordon ZV New Results of Investigations on the Problems of Work Hygiene and the Biological Effects of Radiofrequency Electromagnetic Waves in Biological Effects of Radiofrequency Electromagnetic Fields pp 2 14

- 73. ibid
- 79. ibid
- 80. ibid
- 81. Czerski P et al. Influence of Microwave Radiation on the Hematopoietic System. in; Biologic Effects and Health Hazards of Microwave Radiation WEO International Symposium 1973
- 82. Miro L et al, Effects of Microwaves on the cell metabolism of the reticulohistocytic system. in; Biologic Effects and Hazards of Microwave Radiation WHO International Symposium 1973
- 83. Miro ibid
- 34. Imig C J., Testicular degeneration as a result of Microwave Irradiation Proceedings of the Society for Experimental Biology and Medicine 1948 Vol 69 382 - 386
- 85. Sigier A T et al., Rediction exposure in parents of children with mongolism Bulletin of The John Hopkins Hospital 1965 Vol 117 pp 374 399
- 86. Baranski S et al., Effects of Microwave Irradiation in Vitro on cell membrane permeability, pp. 177 in Biologic effects and health hazards of microwave radiation. Proceedings of an International Symposium Warsaw 1973.
- 87. ibid
- 88. Yao K T S., Cytogenetic consequences of microwave incubation of mammalian cells in culture. Bureau of Radiological Health US Dept HEW 1976
- 88b. Romanov V I., Voprosy primeneniya korotikh i ultrakorotlákh voln v medistina 1940
- 89. Presman A.S., Levitina N.A., Nonthermai Action of microwaves on the rythm of cardiac contractions in animais. Wright Patterson Air Force Base Chio Foreign Technology Division June 1962
- 90. Adey W.R., Electromagnetic fields and the essence of living systems., in, Modern Radio Sciences
- 91. McLaughlin KA and Steiner U. E. Spin correlated radical pair as a reaction intermediary. Molecular Physics Vol 241 pp73 84 1991
- 92. Ulrich T and Steiner U.E. Magnetic field effects in chemical kinetics and related phenomena. Chemical Reviews p 51 89 1989

- 93. McLaughlin AA. Are environmental magnetic fields dangerous. Physics World pp 41 45 Jan 1992
- 94. Illinger K H., Biological efficies of non-ionising radiation, American Chem Soc Symp Ser 157 1981
- 95. Grundler W., et al Mechanisms of Electromagnetic Interaction with cellular systems., Max Planck Institute /1991
- 96. Frolich H., Coherent excitation in active biological systems. in Modern bioelectrochemistry ed F Gutmann and H Keyzer pp 241 61 Plenum Press 1986
- 97. Adey W R op cit
- 93, ibid
- 99. Adey WR. Tissue interactions with non ionising radiation electromagnetic fields. Physiol Rev 1981; 61, 435 514
- 100. Adey W.R., Ionic nonequilibrium phenomena in tissue interactions with non ionising electromagnetic fields. in Biological Effects of non-ionising radiation. Am Chem Soc Symp Serv No 15 1981b
- 101. Adey W.R., Nonlinear, nonequilibrium aspects of electromagnetic field interactions at cell membranes, in Nonlinear electrodynamics in biological systems p. 3-22 Plenum Press 1984
- 102. Adey W.R., The sequence and energetics of cell membrane transductive coupling to intracellular enzyme systems. Biolelectrochem. Bioenerg 1986;15, pp 447-56
- 103. Adey WR., Evidence for tissue interactions with microwave and other non-ionising electromagnetic fields in cancer promotion. in Biophysical Aspects of Cancer. Fiala J Pokorny J eds Prague: Charles University.
- 104. Adey W R., Cell membranes, electromagnetic fields and intercellular communication. in Brain Dynamics; Progress and Perspectives; ed Basar et al Springer Verlag 1987
- 105. Adey W.R., Cell Membranes: the electromagnetic environment and cancer promotion. Neurochem Res., 1983;13, 671-7
- 106. Adey W.R., Physiological signalling across cell membranes and cooperative influences of extremely low frequency electromagnetic fields. In Biological coherence and response to external stimuli.) ed H Frolich pp148-70 Springer Verlag, Heidelberg 1988

- 107. Adey W.R., Biological Effects of Rudiofrequency Radiation. In Interestion of electromagnetic whyer with Biological systems (ed JC Lin) pp 109-140 Plenum 1988
- 103. Adey W.R., Effects of microwaves on calls and molecules. Nature 333; p 401 1988
- 109. Adey W R and Lawrence A F ed; Non linear electrodynamics in biological systems Plenum 1984
- 110. Adey W.R. et al. Effects of week amplitude modulated microwave fields on calcium efflux from awake cut cerebral cortex.

 Bioelectromagnetics 1982 Vol 3 pp 295 305
- 111. Adey W. R. et al, Nonlinear wave mechanisms in interactions between excitable tissue and electromagnetic fields. Neurol Research Vol 4 pp 115-154 1982
- 112. Adey W.R. et al. Effects of weak amplitude modulated microwave fields on clacium efflux from awake cat cerebral cortex. Bioelectromagnetics Vol 3 pp 295 307. 1982
- 113. Grandler W., et al. Phys Left 62a 436 1977
- 114. Grundler et al
- 115. Grundler et al
- 116. Grundler et al , Phys Rev Lett. 51 1214 1983
- 117. Grundler et al., Mechanism of electromagnetic interaction with cellular systems. Max Planck Inst 1992
- 118. Blackman CF et al., Induction of calcium ion efficial from brain tissue by radio frequency radiation: effects of modulation frequency and field strength.

 Radio Science Vol 14 93 -98 1979
- 119. Dutto S K et al., Microwave inducedes leuim efflux from brain tissue in vitro. Bioelectromagnetics, Vol 5 pp 71-78
- 120. Fletcher W.H., et al, A modulated microwave field and fumour promotors similarly enhance the action of alpha lymphotoxin. Proc. Biolectromagnetics Soc; p 12, 8th Annual Meeting 1986
- 121. Sadcikova M N ., The clinic, pathogenesis, treatment, and outcome or radiowave sickness. in Biological Effects of radiofrequency electromagnetic fields ed Gordon 1974

122, ibid

123. ibid

124. New York Workers Compensation Court Records Yannon V New York Telephone Company

125 Adey op cit 1992

126. EPA Evaluation of the Potential Carcinogenicity of Electromagnetic Fields 1990

127. EPA ibid

128. EPA ibid.

129. Prausnitz S and Susskind C. Effects of chronic microwave irradiation on mice. IRE Trans. on Biomed. Electron Vol 9 pp 104 - 108 1962.

130. Szmigielski S. et al. Accelerated Development of spontaneous and benzopyrene-induced skin cancer in mice by microwave radiation. Arch. Dermatol Res. Vol 274 pp 303 - 312, 1982

131. Szmigielski S. et al. Immunologic and cancer related aspects of exposure to low level microwave and radiofrequency fields. in Modern Biolectricity ed Marino pp 861 - 915 1988

132. ibid

132b Robinette C.D. and Silverman C. Causes of Death following occupational exposure to microwave radiation (radar) 1950 - 1974. In Hazzard DG ed Symposium on biological effects and measurements of radiofrequency/microwaves. HEW Publication 77 -8026 pp 338 - 334.

133. Szmigielski S (1988) op cit

134. ibid

135. Microwave News

136. Morton W.E., Radioemission Density and Cancer Epidemiology in the Portland Metropolitan Area. US EPA 1987

137. Environmental Epidemiology Program State of Hawaii 1986

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Potential and Actual Adverse Effects of Radiofrequency and Microwave Radiation at levels near and below 2 μW/cm².

by

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Potential and Actual Adverse Effects of Radiofrequency and Microwave Radiation at levels near and below 2 μW/cm².

by

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1. Background:

The Environment Court (as the Planning Tribunal) in 1995 heard the case of residents against BellSouth, MacIntyre vs BellSouth, and the decision is recorded as Decision A96/15 (MacIntyre Case), NZPT (1996). The decision of the Chief Planning Judge, His Honour Judge Sheppard, was based on the part if the New Zealand Standard NZS 6609 which advocates that exposure should be "as low as reasonably achievable" (ALARA), and on the application of the Precautionary Principle, in conjunction with Sections 5 and 3 of the Resource Management Act 1991 (RMA). The RMA requires that people "avoid, remedy or mitigate any adverse effects of an activity on the environment" (Section 5(2)(c)), including "any cumulative effect which arises over time or in combination with other effects - regardless of scale, intensity, duration, or frequency of the effect, (section 3(d), and also includes" ... "any potential effect of low probability which has a high potential impact" (Section 3(f)).

In respect of the New Zealand Standard the Decision (A96/15) notes:

"However neither that Act (The Standards Act 1988) nor the Resource Management Act gives the New Zealand Standards any status that would bind a consent authority to use them as a basis for deciding a resource consent."

The Tribunal (A96/15) decided that a precautionary approach to the adverse health effects issue was warranted, p 49, :

"We have concluded that the low power density of the proposed transmissions, the condition that we would impose limiting the incident power flux density to 2 μ W/cm² at any dwelling, and the relationship between that limit and the relevant standards referred to in the preceding chapter, all illustrate the application of a precautionary approach."

And in the next paragraph:

"However, this case focused on the possibility of adverse health effects from radiofrequency radiation, and after careful consideration of the evidence we have found that the transmissions would not have any actual or potential adverse effects on the public, not even a potential effect of low probability which has a high potential impact. This can be assured by the amended condition that we would substitute [2 μ W/cm²], and by the provisions of the Act that could be invoked if it should turn out, contrary to

the evidence before us, that the transmissions have an adverse effect, including the ability to review the condition."

The converse of this is that without a condition of $2\mu W/cm^2$, the finding of no actual nor potential adverse effect will not be assured. To underscore and strengthen this position the Tribunal also places an onus on the City Council to monitor compliance with the condition and to review the condition in the light of evidence or adverse health effects near or below $2\mu W/cm^2$. The Resource Consent was granted with the following condition:

"3. That the incident power flux density of radiofrequency radiation emitted by the facility, measured at any dwellinghouse, is not to exceed 2 microwatts per square centimetre."

Hence, while setting the Public Exposure Limit at $2\mu W/cm^2$, the decision requires the review of this condition if there is any new evidence. "New Evidence" includes reevaluation and interpretation of the research presented to the original hearing if sound principles warrant that, as well as the results of studies not presented to the original hearing because of lack of awareness of them or access to them, or they were not published at that time.

2. New Evidence:

There are five sources of new evidence and re-evaluation of four studies. Four new studies include the North Sydney TV tower study of Hocking et al. (1996), the set of papers on the Skrunda Radar in Latvia, the analysis of the effects of the shortwave tower in Schwardenburg, Switzerland, Altpeter et al. (1995) and the Chinese Study of Chiang et al. (1989). These studies all have exposure measurements or calculations associated with them, which increases their power. The study by Dolk et al. (1997a, 1997b) adds confirmation to the Hocking et al. (1996) study. All of these studies show increased risk of adverse effects, on health, well-being and the environment, at mean exposure levels at a fraction of the New Zealand Standard NZS6609 and well below $2\mu \text{W/cm}^2$, as applied in the MacIntyre Case.

Re-evaluation of the U.S. Physiotherapist study, the U.S. Navy Korean War Study, the exposure estimates in the Polish Military Study, and the Von Klitzing EEG study, all of which were presented to the Planning Tribunal in the MacIntyre Case. The first is reinforced by stronger evidence for a mechanism of cumulative cellular damage, the second shows evidence of adverse health effects not presented to the Tribunal, the third shows that exposure hygiene requirements and measurements enable career mean exposure ranges to be estimated, and the fourth is accompanied by collaborative evidence.

The fifth source of new evidence is the improving understanding of the biological mechanisms linking the absorption of electromagnetic radiation into biological tissue, with consequent changes which have the potential or actual effect of causing adverse health effects. This includes resonant absorption of RF/MW radiation at the cell membrane, the pivotal role of calcium ions, the electrobiochemical nature of the molecular structures at the cell surface, the disruption of cell to cell messages, the alteration of the transduction

of messages into cells which affect the cell nature and development, and research showing specific cellular effects under EMR exposure.

2.1 Physical Variables, Units and Formulas Used:

Electromagnetic radiation oscillates electric and magnetic vectors at right angles to each other, traveling at the speed of light, and carrying energy through space. The relationship between the wavelength of the oscillating EM waves, their speed and their frequency is given by:

$$c = f \lambda \tag{1}$$

where c is the speed of light, which in a vacuum is 3 x 10⁸ m/s.

f is the frequency in cycles per second, cps.

λ is the wavelength in m.

EM radiation (EMR) has the characteristics of waves and of particles. Like waves, EMR is reflected, absorbed and transmitted through partial transparent material. It is also diffracted and refracted. The higher the frequency, the shorter the wavelength, the more particle-like EMR becomes. The energy in the wave stream is contained in small packets called quanta. The energy per quantum (E₀) is given by:

$$E_{\alpha} = h f \tag{2}$$

where h is Planck's constant, 6.63 x 10⁻³⁴ J-s.

At any point in the EM wave the electric field (E in V/m) and the magnetic field (H in A/m) are proportional to each other, such that E/H = $120\pi = 377\Omega$, the free space impedance...

The flux of E-M energy which is radiated through space is called the Energy Flux (S), and when it impacts onto an object it is called the Exposure. Exposure relates to the electric field through:

$$S = E^2 / 377 \quad [W/m^2]$$
 (3)

or to the magnetic field:

$$S = 377 \text{ H}^2$$
 [W/m²] (4)

where H is the magnetic field strength in Ampere/m (= A/m) which is related to the Magnetic Flux Density (B) through:

$$B = \mu H \qquad [Tesia] \qquad (5)$$

where μ is the magnetic permeability, $\mu \approx \mu_0 = 1.257 \times 10^{-6}$ H/m (H=Henry).

Exposure is frequently expressed in μW/cm².

$$1 \text{ W/m}^2 = 100 \ \mu\text{W/cm}^2$$
 (6)

As the EM field propagates away from its radiating source (in the far field) the Energy Flux decreases as the square of the radius (r) and:

$$-S = P/4\pi r^2 \tag{7}$$

where P is the total radiated power, and r is the distance from the antenna.

Hence the Exposure of people and objects gets rapidly smaller as distance increases from a transmission facility, such as a radar, a cell site or a TV tower. For each doubling of the distance the Exposure is reduced by one quarter. However, since S varies as the square of electric field and magnetic field, E and H reduce linearly with distance from the antenna in the far field condition.

For an isotropic antenna, radiating equally in all directions, the exposure at any distance from the antenna can be calculated from Eq (7). Almost all commercial antennae have considerable directional focus to send their radiation more intensely in chosen directions so that TV, radio and cell site stations direct their signals towards potential receivers and not out to space. Hence for a given antenna power (P), the exposure at a given radius will be greater than that given by Eq. (7) by factor related to the Antenna Gain (G). The radiation pattern is further complicated by the existence of side-lobes in addition to the primary beam. Hence close to an antenna the exposure pattern is complicated. In the more distant field, outside the influence of the side lobes, the intensity decreases as the inverse square law of Eq.(7), but with a higher initial power determined by the antenna gain characteristics modified by the loss characteristic of the feeds etc. A numerical overall gain factor of 6 to 7 is common for omni-directional antennae on Telecom cell sites.

The Energy Flux which impacts on a object is reflected, scattered and absorbed by the object. The proportion of the energy absorbed is a function of the wavelength of the wave compared to the linear dimension of the object. The most efficient energy absorption occurs when the wavelength of the EM wave is close to twice the size of the object. Larger objects have more efficient energy absorption at longer wavelengths, i.e. at lower frequencies, while smaller objects have higher absorption efficiencies at shorter wavelengths and higher frequencies. A 1.8 m man has a peak absorption rate at about 70 MHz, a monkey at about 300 MHz, an adult nead at about 915 MHz and a mouse at about 2.450 MHz.

On a gross scale the energy absorption is expressed according to the incident absorbed energy's heating ability and it is expressed as a Specific Absorption Rate (SAR), which in terms of the incident electric field is (Ganghi (1990))

$$SAR = \sigma E^2/2 \rho \qquad [W/kg] \qquad (8)$$

where σ is the electrical conductivity of the tissue, in Siemens/m² or S/m², and ρ is the density of the tissue, in kg/m³.

At ELF frequencies electrical conductivities are 0.1-0.35 S/m for cardiac muscle, 0.1-0.3 S/m for nerve tissue, 0.2 S/m for cerebral tissue and 0.25 S/m for myocardial tissue, Repacholi (1993). Typical values for the electrical conductivity of tissue for MW are 0.05 S/m for bone, 0.95 S/m for muscle and 0.77 S/m for visceral organs such as heart, liver, brain etc.

Table 1 : Mid-range a function of			-	for biologi uchly and S		
Tissue	100 kHz	1 MHz	10 MHz	100 MHz	1 GHz	10 GHz
Skeletal Muscle	0.50	0.72	0.83	0.90	1.42	11.5
Liver	0.16	0.28	0.45	0.66	0.98	8.9
Spleen	0.62	0.63	0.67	0.89	1.2	10.1
Kidney	0.25	0.37	0.59	0.86	0.98	9.7
Brain	0.15	0.18	0.42	0.72	1.00	9.1
Bone	0.014	0.017	0.024	0.057		

Density is close to 1000 kg/m³ for most tissue because of the presence of water. However lung tissue is about 100 kg/m³ since it contains pockets of air.

While in terms of the tissue heating rate it is:

$$SAR = C DT/dt$$
 (9)

where C is the specific heat of the tissue, in J/kg-°C, and DT/dt is the rate of increase in tissue temperature (°C/s)

Combining Eq. (3) and Eq. (8),

$$S = (2\rho/3.77\sigma) SAR [\mu W/cm^2]$$
 (10)

and using $\rho = 1000 \text{ kg/m}^3$

$$S = 530.5/\sigma SAR \left[\mu W/cm^2\right]$$
 (11)

Using the data in Table 1 a relationship between σ and frequency has been derived, for example,

for brain tissue:
$$\sigma = 0.27 + 0.0973 \ln (f)$$
 (12)

and for muscle tissue:
$$\sigma = 0.672 + 0.0877 \ln (f)$$
 (13)

where f is the frequency in MHz.

Hence, for example, an SAR of 0.00015 W/kg for isolated frog hearts at 240 MHz (σ = 1.15 S/m) corresponds to exposure of 0.1 μ W/cm² and for Von Klitzing's human brain EEG at 150 MHz (σ =0.76 S/m) and 0.001 W/kg to 0.7 μ W/cm²

The estimate of the SAR for a whole body or body part is a complex calculation because of different tissue densities and electrical conductivities of each tissue type, and the variable size of components of the body which influences the efficiency of absorption of the EMR, all of which varies with wavelength and frequency of the EMR.

Gandhi (1980) gives empirical formulae for the whole body averaged SAR for a 1 mW/cm² exposure as a function of the signal frequency, for when the electric field vector is parallel to the length dimension. Expressing the coefficient in Eq.10 as R (=2p/(3.77 σ) and using the units for R of W/kg per μ W/cm², Gandhi's model is:

Resonant Frequency f = 114/L MHz

$$S_{res} = 15.2 \sqrt{(L^3/m)}$$

For the sub-resonant range: 0.5 f, < f < f,

$$R = 5.2 \times 10^{-3} L^2 / m (f/f_r)^{2.75}$$
 (14)

For the supra-resonant range: $f_r < f < 1.6 S_{res} f_r$

$$R = 0.595 L/(mf)$$

where f is the frequency of the incident signal in MHz.

L is the long dimension in m, and m is the mass of the person in kg.

For example, for an incident signal of 300 MHz, an adult with L=1.8 m and m = 80 kg, f_r = 63 MHz, it is in the supraresonant range and R = 4.44 x 10⁻⁵. For a child with L = 0.9 m and m = 25 kg, f_r = 127 MHz and R = 7.14 x 10⁻⁵. Hence an incident RF signal at 300 MHz and power density of 20 μ W/cm² would produce an SAR for the adult of 0.0009 W/kg and the child of 0.00179 W/kg, two times higher for the child than the adult. This ratio remains the same for all frequencies since it is determined by the L/m ratio.

Figure 1 shows the calculated SAR levels produced by a content energy flux of 1 mW/cm² as a function of frequency, for various body sizes.

When an electric field is induced in a person by the incident RF signal, an electric current flows through the person to earth. Gandhi (1990) has shown that the electric current which is flowing through the feet of a grounded man (I_h in mA) as the result of an incident electric field E (V/m) is given by:

$$l_h = 0.108 E h_m^2 f$$
 (15)

For a 1.75m person at 40 MHz, at the limit field exposure for the ANSI C95.1 safety standard, 63.2 V/m (1 mW/cm²) this gives a current of 836 mA.

The localized SAR is a function of I_h , which flows down two legs and since SAR= $J^2/\sigma\rho$,

SAR =
$$(I_{\rm h}/2)^2/(A_{\rm e}^2 \sigma_{\rm c} \rho)$$
 (16)

where J is the current density (A/m^2) , A_e is the effective cross sectional area of the legs, at the ankles it is about 9.5 cm² even though the physical cross section is about 40 cm².

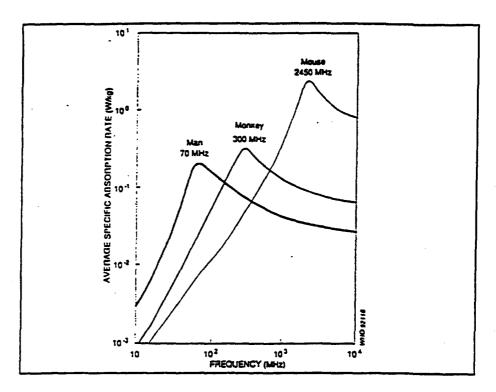


Figure 1: The average SAR for 3 species exposed to 1 mW/cm² with the E vector parallel to the long axis of the body, Durney et al. (1978).

Hence, assuming a density of 1000 kg/m³ and σ_c =0.77 S/m, this person has an SAR at their ankles of 251 W/kg. The ANSI standard is based on thermal protection and states that SAR should not exceed 8 W/kg for any 1 g of tissue in occupational exposures and 1.6 W/kg for public exposures. Since the exposure S is proportional to SAR, to protect a well grounded adult person from heat exceeding 1.6 W/kg in the ankles the limit exposure near the resonant frequency would need to be reduced to 6.4 μ W/cm². Insulating people with rubber soled shoes for example, reduces the induced current by 60 to 80 %. However, very young children frequently play with bare feet and so can often be in a well grounded state.

2.2 Thermal limits for young children:

An exposure of 1 mW/cm² (whole body mean SAR=0.4 W/kg), for a 10 yr old child (h = 1.38m, f = 50.7 MHz, A_e =6.1 cm²) SAR = 371 W/kg; for a 5 yr old child (h = 1.12 m, f = 62.5 MHz, A_e = 4.2 cm²) SAR = 534 W/kg (Gandhi (1990) and for a 2.5 yr old child (h= 0.9 m, f = 74 MHz, A_e = 3.0 cm²) SAR = 603 W/kg. Hence, using simple ratios since S is directly proportional to SAR, for children the allowable exposure to limit the heating of any 1 g of tissue to 1.6 W/kg is 4.2 μ W/cm² for a 10 yr old, 3.0 μ W/cm² for a 5 year old and 2.65 μ W/cm² for a 2.5 yr old.

Professor Gandhi notes that an empirical fit to heating rate as a function of SAR yields 0.0045 X SAR (W/kg) °C/min. If a 2.5 yr old toddler is exposed to 1 mW/cm², then the heating rate will be 2.73 °C/min. Prof Gandhi concludes that for the ankle section "substantial rates of surface temperature elevation are anticipated."

2.3 Standards give inadequate protection even for thermal effects in children:

The Australian and New Zealand joint standard does not protect from these localized heating effects through its use of electric field intensity and related exposure limits based on whole body average SARs only, allowing 0.4 W/kg for occupational exposure and 0.08W/kg for public exposure.

Even at our lower public exposure level of 0.08 W/kg maximum heating rates in toddler's ankles is about 0.55° C/min. This has serious implications when children are exposed to FM signals in the frequency band 20 to 150 MHz especially. Since the public exposure standard must be adequate to protect even the most vulnerable, if they are set to only deal with thermal effects, then in the sub-150 MHz range they should be adequate to protect a baby from adverse heat levels. This would require the allowed maximum level to be set at $2 \, \mu$ W/cm² or less, simply to meet the 1.6 W/kg limit.

These limits will be somewhat higher away from the optimal frequency for maximum induced current flow, but these figures illustrate the vulnerability of body parts to high levels of localized heating and to the greater vulnerability of young children.

While short-term exposures can give dangerous heating effects down to exposure levels far below the current "safety standard" when near the absorption frequency maximum, legitimate concerns exist about changes in our brains and at the cellular level the short-term exposures might cause. There is also clear evidence about chronic exposures at far lower levels of exposure which have the potential to alter the ambient electromagnetic environment in ways which are potentially harmful through effects on reproductive processes, brain function and metabolism, sleep disruption, immune system suppression and EMR probably causes an increase in the risk of cancer.

2.4 Natural Electromagnetic Environment:

The earth's static magnetic field is about 30-50 μ T and static electric field (in fair weather conditions) is about 150 V/m. Our bodies are well adapted to these static fields and to the radiation from the sun. We are now seeing how small changes to a minute part of the solar spectrum, UVA and UVB, are producing significant increases of skin cancer in non-black skinned people. Legitimate concern can be raised about the significant increases in population exposures to other parts of the EM spectrum from ELFs to millimeter microwaves.

Our background thermal environment emits around 400 W/m² (40,000 μ W/cm²) of "thermal" radiation, the vast majority of which is in the infrared. The part of this which forms the natural sources of oscillating EMR fields summed over the range up to 300 GHz is 0.3 μ W/cm², Repacholi (1983). In the radiofrequency range it is less than 10 μ W/cm² because over 99.996 % of the 0.3 μ W/cm² comes from above 10 GHz, whereas most of our spectrum use for telecommunications, (radio, TV, RT and cell phones, satellite communication, radar) are in specific frequency bands below this frequency. Hence from the ELF to RF/MW parts of the EM spectrum we have massively increased our ambient exposure, especially following developments since the Second World War.

Figure 2 shows how a cell site local exposure level of 2 μW/cm² stands out against the

The ionosphere has a net positive charge which creates a static electric field and the dynamics of thermal convection in the earth's core produce the earth's magnetic field. Thunderstorms create short-term localized field variations as they strongly alter the local electric field and lightning produces emissions broad spectrum emissions of RF/MW in bursts called sferics. These can be heard on radios as a burst of static.

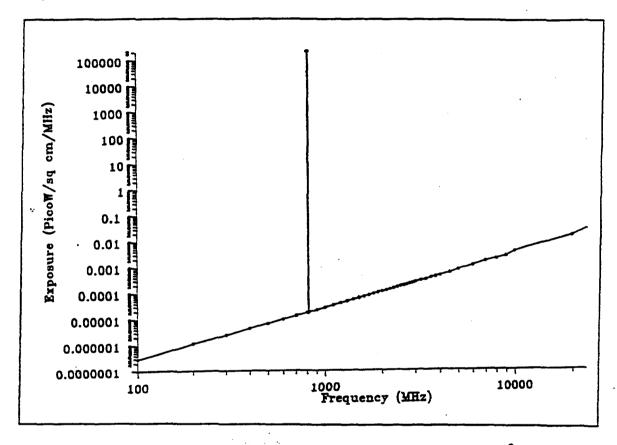


Figure 2: The signal of a typical cell site near the mast (2μW/cm²) against the thermal radiation background in the frequency range around that used by cell sites.

Long-term natural ELF signals arise from thunderstorm sourced electromagnetic energy being ducted around the world in the cavity between the earth and the ionosphere. These are called the Schumann Oscillations. Their fundamental frequency is 7.8 Hz, with harmonics at 14.1, 20.3, 26.4 and 32.5 Hz. Their amplitude is about 0.2 mV/m for a 1 Hz bandwidth, Campbell (1967). Assuming each resonant frequency is associated with a 1 Hz bandwidth, the total field strength will be 1 mV/m or 0.1 V/cm. From Eq. 3 this is equivalent to an energy flux of 0.27 pW/cm².

2.5 Modification of the natural environment:

While life on earth is well adapted to the static magnetic and electric fields and to minute intensities of naturally occurring ELF and RF/MW fields primarily from thunderstorms, biological tissues and organisms have, in this century, become exposed to ELF, RF/MW (VHF and UHF) fields at intensities which are thousands of times higher than they were at the turn of the century (<10pW/cm²). The median public RF exposure measured by the U.S.E.P.A. in U.S. cities in 1979 was 0.005µW/cm² or 5,000 pW/cm², Tell and

Mantiply (1980). About 1 % of the population lived in more than 1μ W/cm² or 1 million pW/cm².

2.6 Healthy vs vuinerable people:

People who live or work close to transmission facilities have mean daily exposures of about 1 to $20\mu \text{W/cm}^2$, but when averaged over a year their mean is of the order of 0.2 to 5 $\mu \text{W/cm}^2$. In the work and military environment a "healthy worker" effect exists employment selection screens out the sick, young and elderly. Hence while occupational and military exposures can be somewhat greater than average in certain cases, studies on these workers tend to significantly underestimate the effect similar exposures would have on the vulnerable in society. It is the vulnerable groups in which overall sickness rates are higher. So when reviewing national morbidity and mortality statistics the vulnerable groups are over represented and there are large groups of "healthier than average" people who are under-represented in the health statistics.

The IRPA RF exposure standard is an acknowledged thermal standard based on an occupational exposure limit of 0.4 W/kg. Greater protection is offered to the general public because this includes the vulnerable. Hence the Public Exposure Limit of 0.08 W/kg, which will avoid burns, and slight heating effects in infants and the frail elderly on hot sunny days when they exercise, Repacholi (1993). However such standards do not deal well with hot spots, nor with potential nor actual health effects from chronic exposure.

3. Absorption Mechanisms:

Just as a radio or television can detect and decode modulated RF/MW signals at RMS intensities at a minute fraction of the static electric and magnetic fields, using frequency banded tuned oscillators, so can biological organisms absorb and resonate in modulated RF/MW fields. Human and animals bodies act as antennae for whole body energy absorption and tissue cells act as resonant absorbers. The whole body absorption determines the energy absorbed, the fields induced and the currents which flow through the body. Many fundamental cellular processes involve electric fields and the flow of ions. Hence induced changes from imposed EM fields can and do alter the cellular processes and their ionic balance.

3.1 Biological responses:

On the macro-scale, human and animal circadian rhythms are driven by the day/night cycle with a phase-lock synchronization provided by environmental ELF fields (E<0.3 pW/cm²). A fundamental physiological aspect of the circadian rhythm involves the pineal gland and the secretion of a neurohormone called melatonin. Light falling on the eye's retina produces signals which are biochemically amplified around a million times, to stimulate the pineal gland to reduce its melatonin output.

3.1.1 Pineal Melatonin - A plausible mechanism for EMR effects

Pineal and serum melatonin concentration drops during the day and rises overnight, Figure 4. Melatonin production is very well understood. A schematic of the way in which

many molecules of the neurotransmitter, norephinephrine (NE), which is received by receptors on the surface of the pineal gland cell (Pinealocyte). Tryptophan is converted to serotonin which is then converted to Melatonin at a rate controlled by an enzyme NAT (N-acetyltransferase) which has been activated or limited through protein synthesis from amino acids controlled by cyclic AMP, Figure 3. The melatonin easily passes through the cell wall into the blood stream to be dispersed throughout the body.

The pineal gland is located near the centre of the brain. It is an endorcrine organ which produces most of the melatonin which is found in the blood, figure 5.

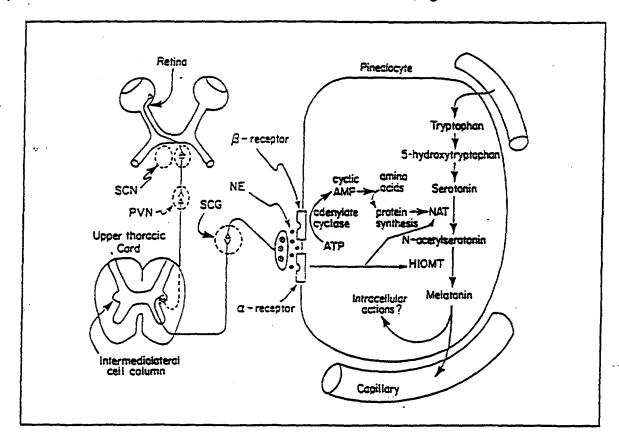


Figure 3: The connection of the eyes (retina) to the pineal gland, represented by a single pinealocyte, and the synthesis of melatonin within the gland. Tryptophan, an amino acid from the blood, is converted to the hormone melatonin, which is quickly released into the capillaries of the gland. The enzymes which catalyze the conversion of serotonin to melatonin include Nacetyltransferase (NAT) and hydroxyindole-O-methyltransferase (HIOMT). The pineal gland produces melatonin at night since the nerve endings which end in the pineal gland release the neurotransmitter norephinephrine (NE) which interacts with the b- and a-adrenergic receptors on the cell membrane; these interactions initiate the processes which control melatonin production. ATP. adenosine triphosphate; PVN paraventricular nuclei: SCN. suprachiasmatic nuclei; SCG, superior cervical ganglia.

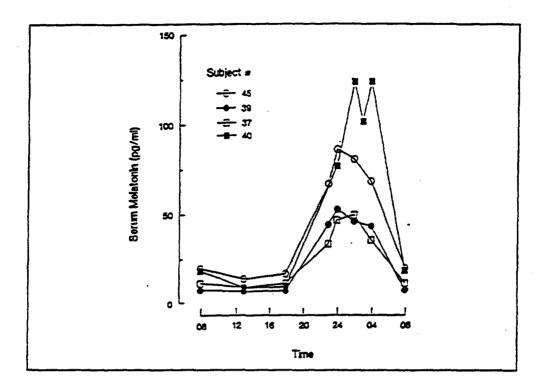


Figure 4: Blood melatonin levels for 4 adult males over a 24 h period, Reiter (1994).

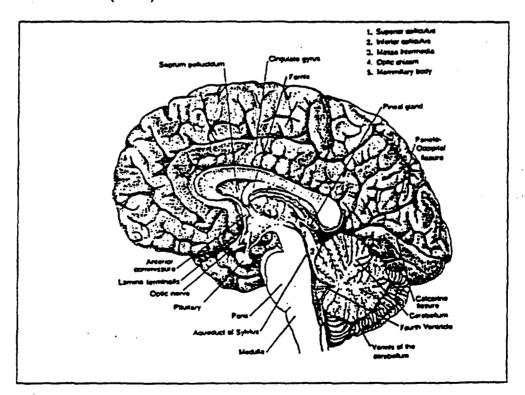


Figure 5: A mid-saggital section of the human brain showing the location of the pineal gland, Reiter (1994).

Once melatonin is produced it is the molecule's high ability to pass through the cell membrane which allows to escape from the pinealocyte to the blood. Once in the blood melatonin has access to every cell in the body, which is passes through the cell membranes where every nucleus has receptors for it. A few cell membranes have

receptors. These may mediate the 24 h circadian rhythms of the endocrine system. In the nucleus melatonin plays a role in regulating the effects of the indole on gene expression. The ability of melatonin to enter all cells is also essential for one of the other important functions of melatonin, namely, its ability to scavenge the highly toxic hydroyl radical (•OH).

The production of oxygen-based free radicals, such as •OH is a consequence of the utilization of oxygen by organisms. About 1-2 % of inspired oxygen ends up as toxic free radicals. It is generally considered the •OH, because of its high reactivity, is the most devastating to macromolecules such as DNA, proteins and lipids. The cellular damage produced by free radicals is generally referred to as oxidative stress, Reiter (1994).

Because of its action in removing free radicals, melatonin is probably the most efficient natural cell protection and oncostatic agent in our bodies. Every night, our pineal produces large quantities of melatonin which flood almost every cell in our body, cleaning out the free radicals and assisting cell division to take place with undamaged DNA. As we age our nocturnal peak melatonin production falls markedly, making elderly people much more prone to cancer.

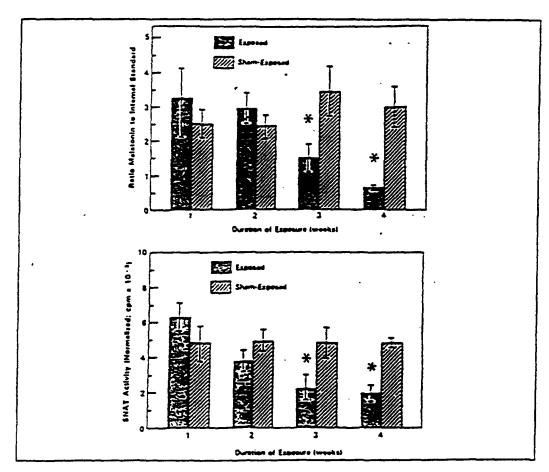


Figure 6: Pineal melatonin (top) and NAT activity (bottom) in groups of rates exposed to a modulated electric field for 1 to 4 weeks. The glands of the animals were collected at night. In the sham-exposed animals the pineal melatonin and NAT levels were always high. However, after both 3 and 4 weeks of exposure to the electric field, both parameters were depressed (p<0.001).

To test the cancer protecting properties of melatonin, Tan et al. (1993), injected rats with a chemical carcinogen, safrole. Safrole normally damages DNA because it induces the production of large numbers of free radicals. Rats injected with Safrole were found to have extensive DNA damage after 24 h. When melatonin was also injected, the DNA damage was reduced by 99 %. Since damaged DNA can undergo mutation it may result in the growth of a tumour. Thus melatonin is clearly a potent cellular protector against cancer initiation.

Three independent laboratories, Battelle PNL (Wilson), U.C. Riverside (Luben) and the U.S. EPA (Blackman), have shown that 60 Hz modulated magnetic fields in the 1 to 12 mG range, almost completely negate the oncostatic effect of melatonin in human breast cancer cells, with a dose-response relationship. Wilson et al. (1986) showed significant reductions in pineal melatonin in living rats when they were chronically exposed to 60 Hz modulated electric field at 1.7-1.9 kW/m for 20 h per day, for 30 days. The results are shown in figure 6.

Lerchl et al. (1988) samples for serotonin and its derivatives by periodically inverting the magnetic field at night. Figure 7.

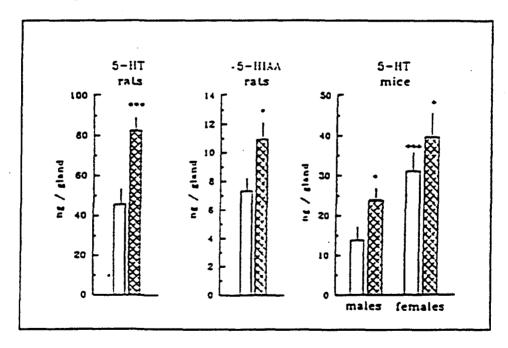


Figure 7: Pineal serotonin (5-HT) and 5-hydroxyindole acetic acid (5-HIAA) levels in rats and mice (cross-hatched bars) with and without (clear bars) exposure to pulsed static MF at night. Both 5-HT and 5-HIAA levels increased as a result of the exposure; these changes are consistent with a reduced melatonin production. • p<0.05 and *** p<0.001 vs control; +++ p<0.05 vs control male mice, from Lerchl et al. (1988).

This review paper by Professor Russell Reiter was prompted by a number of epidemiological studies in which an increased incidence of cancer was reported in individuals living or working in an environment of higher than normal artificial electromagnetic fields. Because of the key role of melatonin is decreasing the likelihood of cancer because of its effect of removing free radicals, Prof. Reiter has been researching the effects of EM fields on melatonin production. The paper abstract concludes with the following observation:

"Reduction of melatonin at night, by any means, increases cell's vulnerability to alteration by carcinogenic agents. Thus, if in fact artificial electromagnetic field exposure increases the incidence of cancer in humans, a plausible mechanism could involve a reduction in melatonin which is a consequence of such exposures."

He also notes:

"Epidemiologists should look for other possible changes, including psychological depression, fatigue, sleep inefficiency, chronic feelings of jet lag, endocrine disturbances and other symptoms; all these may result from a chronically low melatonin rhythm."

Hence there exists a plausible mechanism for cancer and a host of disorders, most of which will be identified below as discovered in epidemiological studies. Often the papers or reports which identify statistically significant increases in cancer or other complains associated with above average exposure to EM fields, have rather weak conclusion, citing the lack of a plausible mechanism. In fact their conclusions can be much stronger because of the existence of the melatonin mechanism and several others which will be described.

Dr Reiter's review paper, quoted above, demonstrates the fundamental role of cells and the vast amount of cellular biochemistry which is known. It also documents biological mechanisms which are chemical and biochemical and <u>are definitely not thermal</u>.

3.1.2 Hypothesis for modulated RF/MW effects on melatonin:

Since it has been shown:

- That ELF magnetic fields do reduce melatonin production in living rats brains;
- That RF/MW signals produce tissue level electric fields about a million times higher than imposed ELF signals, Adey (1981);
- That RF/MW signals are resonantly absorbed at the cell membrane, Liu and Cleary (1995);
- That altering the electric and thermal fields on the surface of the cell membrane change the bining characteristics of H⁺ and Ca⁺⁺ ions on the outer surface of the membrane:
- That modulated RF/MW has been shown to induce significant calcium ion efflux from cells:
- That it known that the cyclic AMP signal transduction pathway and the Calcium ion signal transduction pathway interact; and
- That in the pinealocyte cell the cAMP pathway assists in regulating the transformation of serotonin to melatonin: